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A prospective randomized, controlled, double blinded study to compare the analgesic efficacy of lidocaine (3 mg/ kg 0.5%) with dexmedetomidine (0.5 mcg / kg) and lidocaine (3mg/ kg 0.5%) with normal saline for intravenous regional anesthesia in patients posted for upper limb surgeries

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Abstract
Intravenous regional anaesthesia may be defined as reversible state of anaesthesia, produced by administration of local anesthetics into the venous system of upper and lower extremities. This technique is simple, effective, cheap and quite safe for operations on limbs especially in emergency situations. The present study involves the comparison of analgesic efficacy of Lidocaine with normal saline vs lidocaine with dexmedetomidine in intravenous regional anesthesia· A total of 120 patients belonging to both sexes of ASA – I or ASA – II and between the age groups of 18 to 50 yrs, admitted for various orthopaedic surgeries on forearm were included in the study. They are prospectively randomised in to two groups: Group L-Lidocaine 3mg/kg 0.5%+1 ml normal saline diluted with normal saline made to 40 ml and Group LD –Lidocaine 3mg/kg0.5%+Dexmedetomidine 0.5µg/kg (diluted to 1ml) diluted with normal saline made to 40 ml. Results analysed and discussed. The addition of 0.5 µg/kg dexmedetomidine to lidocaine for intra venous regional anaesthesia has significant analgesic effect. It shortens the onset of sensory and motor block onset, improved tourniquet tolerance and prolonged postoperative analgesia with stable cardiorespiratory parameters.

Keywords: randomized, controlled, double blinded, lidocaine, dexmedetomidine

Introduction
Intravenous regional anaesthesia may be defined as reversible state of anaesthesia, produced by administration of local anesthetics into the venous system of upper and lower extremities. This technique is simple, effective, cheap and quite safe for operations on limbs especially in emergency situations. For example in patients with full stomach and multiple injuries intravenous regional anaesthesia is often a safer option than general anesthesia, particularly if the patient is elderly, or has cardiovascular or respiratory disease. The commonly used local anaesthetic solutions are Lignocaine, mepivacaine and Prilocaine [1]. Theoretically, 2-chloroprocaine should be an ideal agent for Intra venous regional anaesthesia because it is so rapidly bio transformed [2]. Unfortunately, the high incidence of thrombophlebitis reported when this agent was used led to abandoning this agent for Intra venous regional anaesthesia. Prilocaine is another agent that is attractive because of its low toxicity. The possibility of methaemoglobinaemia has discouraged anaesthetists from using this agent, even though Mazze and others have demonstrated that methaemoglobinaemia was not a problem when prilocaine was used for Intra venous regional anaesthesia [3]. Several LA adjuvants have been attempted with variable degrees of success but their use was limited by side effects.eg mivacurium, opioids. α-2-Adrenergic receptor (adrenoceptor) agonists have been the focus of interest for their sedative, analgesic, and perioperative sympatholytic and cardiovascular stabilizing effects with reduced anaesthetic requirements [4-6]. Dexmedetomidine, a potent α-2-adrenoceptor agonist, is approximately 8 times more selective towards α-2adrenoceptors than clonidine [6-7]. Dexmedetomidine has been shown to decrease anaesthetic requirements by up to 90%. The present study involves the comparison of analgesic efficacy of Lidocaine

with normal saline vs lidocaine with dexmedetomidine in intravenous regional anesthesia [8-11].

Methodology

A total of 120 patients belonging to both sexes of ASA – I or ASA – II and between the age groups of 18 to 50 yrs, admitted for various orthopaedic surgeries on forearm were included in the study. Study conducted in government general hospital, Kakinda from January 2012 to July 2013.They are prospectively randomised in to two groups: Group L-Lidocaine 3mg/kg 0.5%+1 ml normal saline diluted with normal saline made to 40 ml and Group LD – Lidocaine 3mg/kg0.5%+Dexmedetomidine 0.5µg/kg (diluted to 1ml) diluted with normal saline made to 40 ml. Patients with known hypersensitivity to local anaesthetics, known epilepsy disorder, prior neurological or vascular injury and crush injury of limb are excluded from the study. All the patients are subjected to test dose of local anaesthetic drug, one hour before starting the procedure.

Demographic profile of the patients were documented. All patients received, midazolam 0.15 mg/kg and atropine 0.01 mg/kg were administered 45 min before surgical procedure. Intravenous regional anaesthesia was achieved using

- Group L-Lidocaine 3mg/kg 0.5% + 1ml normal saline diluted with normal saline made to 40 ml.
- Group LD–Lidocaine 3mg/kg 0.5% + Dexmedetomidine 0.5µg/kg (diluted to 1ml) diluted with normal saline made to 40 ml

The solution was injected over 90sec by an anaesthesiologist blinded to the injected drugs. The sensory block was assessed by a pinprick performed with a 22-gauge short-bevelled needle and taken out continuously every 30 seconds. Patient response was evaluated in the dermatomal sensory distribution of the medial and lateral ante brachial cutaneous, ulnar, median, and radial nerves. Motor function was assessed by asking the subject to flex and extend his/her wrist and fingers, and complete motor block was noted when no voluntary movement was possible. Sensory block onset time was noted as the time elapsed from injection of study drug to sensory block achieved in all dermatomes, and motor block onset time was the time elapsed from injection of study drug to complete motor block. After sensory and motor block was achieved, the distal cuff was inflated to 60 mm Hg above patient's systolic blood pressure followed by release of the proximal tourniquet and the operation was then started. Intraoperatively, boluses of 1µg/kg fentanyl were provided for tourniquet pain treatment when required (when visual analogue scale was >3), Through the operation period if no tourniquet pain was encountered, the beginning of tourniquet pain was accepted as the duration of tourniquet application time Sedation measured by Wilsons grading intraoperatively every 10 min after the injection anaesthetic and postoperative period 30 min, 2hr, 4hr, 6hr, 8 hr, 12hr, 24hr.

Grading of sedation was evaluated by a Wilson's sedation scale

1 Fully awake & oriented
2 2Drowsy
3 3Eyes closed but arousable to commands
4 4Eyes closed but arousable to mild physical stimulus
5 5Eyes closed but not arousable to mild physical stimulus

The tourniquet was not deflated before 30 min and was not inflated for 1.5 h. At the end of surgery, the tourniquet deflation was performed by cyclic deflation technique. Sensory recovery time was noted (time elapsed after tourniquet deflation up to recovery of pain in all dermatomes determined by pinprick test). Motor block recovery time was noted. After the tourniquet deflation, at 30 min, and 2, 4, 6, 12, and 24 h, hemodynamic variables, pain and sedation values, time to first analgesic requirement, and side effects were noted. Patients were given 75 mg IM diclofenac when VAS was >3. The duration of analgesia was the time that elapsed between tourniquet release and the first intra muscular intake of diclofenac as analgesic.

Results

A total of 120 patients were included in the study of which mean age was 35.07 years in Group L and 31.87 years in Group LD. There was no significant sex predilection comparatively among both the groups. Mean weight in group L was 56.73±5.2 kg and in group LD was 56.4±4.8 kg. Duration of surgery comparable (L = 57.03 min and LD = 55.27 min) in both the groups with p value 0.357 statistically not significant. The mean time of onset of sensory blockade was 5.3±1.03 min in group L and 4.40±1.09 min in group LD. The statistical analysis by unpaired t test showed statistically significant difference (p=0.0018) between the two groups. The mean time of onset of sensory of motor blockade was 11.06±1.47 min in group L and 9.93±1.23 min in group LD. The statistical analysis by unpaired t test showed statistically significant difference (p 0.002) between the two groups. The mean time of sensory block recovery time was 4.71±1.04 min in group L and 7.73±2.03 min in group LD. The mean time of motor block recovery time was 5.65±1.01min in group L and 8.5±1.76min in group LD.

Mean onset of tourniquet pain was 49.7±6.27 min in group L and 63.1±6.73 min in group LD. Throughout the operation period if no tourniquet pain was encountered, the beginning of tourniquet pain was accepted as the duration of tourniquet application time. The mean duration of analgesia was 54.33±8.07 min in group L and 406.8 ± 65.35 min in group LD. The statistical analysis by unpaired t test showed statistically significant difference (p<0.0001) between the two groups. Intra operative haemodynamics is compared in table 1.

Table 1: Haemodynamics during the procedure as compared in both the groups.

	Systolic Blood pressure		Diastolic Blood pressure		Pulse Rate	
minutes	**Group L**	**Group LD**	**Group L**	**Group LD**	**Group L**	**Group LD**
0 min	125.00±14.56	123.00±12.64	77.67±8.58	79.33±7.85	83.85±10.32	83.47±8.29
5 min	119.00±11.55	118.33±11.47	75.33±8.19	72.00±7.61	77.60±10.75	75.80±10.12
10 min	118.67±11.06	115.33±9.00	74.00±8.94	73.33±8.02	77.57±7.98	79.13±11.67
15 min	117.00±11.19	114.33±8.98	73.67±8.09	70.67±7.40	77.30±7.22	78.97±10.08
20 min	121.67±12.89	119.33±10.81	76.00±9.32	76.67±8.84	79.27±6.88	78.80±8.81

25 min	117.33±12.02	119.67±10.98	75.33±8.60	73.33±8.02	76.69±7.72	79.43±9.55
30 min	116.00±12.21	114.00±12.21	72.33±7.74	74.0±8.94	75.20±7.51	78.40±5.37
45 min	117.00±12.64	116.67±10.28	71.33±8.19	73.33±8.02	78.63±6.46	81.33±7.54
After tourniquet deflation	111.00±12.96	109.00±10.29	71.67±7.91	70.00±7.43	78.07±5.15	78.00±6.20

In group L 46 patients has sedation score of 1 and in group LD 38 patients has sedation score of 1. It was statistically insignificant with p value greater than 0.05. Mean score of 2 was observed in 14 patients in group L and 22 patients in group LD statistically insignificant. Side effects documented in both the groups are charted in table 2.

Table 2: Side effects in both the groups.

Side effects	Group L (N=30)	Group LD (N=30)	P value
Hypotension	(3%)	(6%)	0.4977
Bradycardia	(7%)	(10%)	0.6133
Nausea	(3%)	(6%)	0.4977
Vomitting	0	0	-
Shivering	0	0	-
Respiratory depression	0	0	-

Discussion

The pharmacologic properties of α-2 agonists have been extensively studied and have been employed clinically to achieve the desired effects in regional anaesthesia. Dexmedetomidine is a highly selective α_2 -adrenoreceptor agonist approved as intravenous sedative and adjuvant to anesthesia. Dexmedetomidine when used intravenously during anesthesia reduces opioid and inhalational anesthetics requirements. Addition of α-2 agonists to local anaesthetics resulted in faster onset of action of local anaesthetics, rapid establishment of both sensory and motor blockade, prolonged duration of analgesia into the post-operative period, dose-sparing action of local anaesthetics and stable cardiovascular parameters makes these agents a very effective adjuvant in regional anaesthesia. Indeed the addition of clonidine during intravenous regional anaesthesia was shown to improve tourniquet pain but did not influence the speed or quality of intravenous regional anaesthesia. (Gentili *et al.*, 1999) [12].Its effect on prolonging postoperative analgesia controversial. (Gentili *et al.*, 1999)[12].

Dexmedetomidine a potent α-2 adrenoceptor, 8 times more selective towards α-2 adrenoceptors than clonidine. Dexmedetomidine –Lidocaine has been used to provide intravenous regional anesthesia and was shown to improve quality of anaesthesia, tourniquet pain and postoperative analgesic requirement. (Memis *et al.* 2004 [13], Esmaglou *et al.* 2005) [14]. these reports suggest that Dexmedtomidine a better adjuvant to lignocaine in providing Intravenous regional anaesthesia.

In a study conducted by Dilek memis, Alparslan Turan *et al.* addition of Dexmedetomidine (0.5µg/kg) to Lidocaine 3mg/kg 0.5% resulted in earlier onset of sensory blockade and motor blockade compared to placebo significant with a p value less than 0.05 [13].

In a study conducted by Yasser M. Nasr, Salwa H. Waly *et al.* addition of dexmedetomidine (1µg/kg) as an adjuvant to lignocaine resulted in earlier onset of sensory and motor blockade compared to control and tramadol group15. The mean time of sensory block recovery time was 4.71±1.04 min in group L and 7.73±2.03 min in group LD. The mean time of motor block recovery time was 5.65±1.01min in group L and 8.5±1.76 min in group LD. The statistical analysis by unpaired t test showed statistically significant difference (p<0.0001) between the two groups. Similar results were shown in the study by Dilek Memis, Alparslan Turan *et al.* sensory block recovery time was 7±3 min in study group (dexmedetomidine0.5µg/kg) as an adjuvant to lignocaine and 4±1 min in the control group[13]. Motor block recovery time was 8±3 min in the study group and 5±1 min the control group. Sensory and motor block recovery times were also statistically significant (P<0.001).

Yasser M. Nasr, Salwa H. Waly *et al.* concluded that addition of dexmedetomidine 1µg/kg resulted in prolonged sensory and motor block recovery time than control group and tramadol group [15]. In our study we observed improved tourniquet tolerance in group LD. Mean onset of tourniquet pain was 49.7±6.27 min in group L and 63.1±6.73 min in group LD. The statistical analysis by unpaired t test showed statistically significant difference (p<0.001) between the two groups. Similar results were shown in the study conducted by Dilek Memis, Alparslan Turan *et al.* [13] and in the study conducted by Yasser M. Nasr, Salwa H. Waly *et al.*, [15] compared with control group with p values less than 0.001 statistically significant.

Dr. Mounis A Abosedira compared the effect of adding Clonidine 1µg/kg and Dexmedetomine1µg/kg for Lidocaine 0.5% for intravenous regional anesthesia [16]. They observed significantly lower VAS scores in intraoperative period for tourniquet pain in Dexmedetomidine group. In our study mean duration of analgesia was 54.33±8.07 min in group L and 406.8±65.35 min in group LD. The statistical analysis by unpaired t test showed statistically significant difference (p<0.0001) between the two groups. VAS scores were statistically significant in first 6 hours.

Dilek Memis, Alparslan Turan *et al.* conducted a study adding dexmedetomidine 0.5µg/kg to lignocaine prolonged the duration of analgesia 564 ±144 min compared to control group 129 ± 54 min [13]. It was statistically significant with p value less than 0.001. In this study they mentioned that statistically significant VAS scores in first 6 hours similar to our study. Yasser M. Nasr, Salwa H. Waly *et al.* conducted a study adding dexmedetomidine1µg/kg and tamadol 100 mg to lignocaine prolonged the duration of analgesia compared to control group and tramadol group [15].

In the study conducted by Dr Mounis an Abosedira [16] observed that there is significant reduction in analgesic consumption both intraoperative and early postoperative period in dexmedetomidine group compared to control group. Peripherallyα-2 agonists produce analgesia by reducing release of nor epinephrine. Or causing α-2 receptor independent inhibitory effect on nerve fibres action potential. (Gaumann *et al.* 1992) [17]. in our study there was no statistical difference between the groups for sedation values both in intraoperative period and postoperative period. Similar resuts were observed in the study conducted by Dilek memis, Alparslan Turan *et al.* [13] They conducted a study by adding Dexmedetomidine 0.5µg/kg to Lidocaine 3mg/kg 0.5% similar to our study. Yasser M. Nasr, Salwa H. Waly, Dr Mounis A Abosedira conducted a study adding 1µg/kg of dexmedetomidine to Lidocaine for intravenous

regional anesthesia [14-15]. They observed significant sedation scores when compared to control group. In our study the intra operative haemodynamic variables were comparable in both the groups. 7% of patients in group L, 10% in group LD had bradycardia. 3% of patients in group L, 6% in group LD had hypotension and nausea. These values are statistically not significant with p value greater than 0.05. None of the patients in two groups had any other side effects like respiratory depression, shivering, vomiting etc.

Conclusion

Intravenous regional anaesthesia is technically simple and reliable but limited by tourniquet pain and the inability to provide postoperative analgesia. Adjuvants will improve tourniquet tolerance and provide prolonged postoperative analgesia. We conclude that the addition of 0.5 µg/kg dexmedetomidine to lidocaine for intra venous regional anaesthesia has significant analgesic effect. It shortens the onset of sensory and motor block onset, improved tourniquet tolerance and prolonged postoperative analgesia with stable cardiorespiratory parameters.

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